Drugs that strongly inhibit prostaglandin synthesis do not reduce the glomerular filtration rate or sodium excretion in hypertensive patients.[5] In contrast, acute nephropathy requiring peritoneal dialysis occurred in a patient with compensated congestive heart failure treated with indomethacin.[2] Renal function deteriorated considerably after treatment with piroxicam in both our patients, which indicates a hitherto unrecognised adverse reaction to this drug. Since piroxicam seems a promising treatment for rheumatoid arthritis this possible association of prolonged treatment with Henoch-Schönlein purpura must be confirmed or refuted.

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Rebreathing aborts migraine attacks

Four patients have been described[1] who hyperventilated during their migraine attacks, which made their symptoms worse and in one case produced carpopedal spasm. There have been further reports (personal communications) in which one subject produced an attack by blowing up balloons, and another, trying to assuage the symptoms of hyperventilation including carpopedal spasm which accompanied some of her attacks, found that rebreathing also abolished the nausea and reduced the headache. I describe six patients who were able to abort many of their attacks by rebreathing their own expired air from a bag.

Case reports

Case 1—A 37-year-old accountant with a history of classical migraine since childhood presented at this migraine clinic at the onset of an attack. He had had a visual aura of patchy scotomata for approximately 20 minutes before presentation and parathesiae affecting his fingers. His aura usually lasted 40 minutes and would then be followed by nausea, sometimes vomiting, and headache lasting roughly six hours. Examination showed a left-sided ptosis which the patient had had since his early teens. Otherwise there were no findings of note. The patient was asked to breathe through a nylon mouth valve into a bag, 25 cm × 20 cm made from laminated polyethylene and polyethylene terephalate, a combination of material resistant to highly diffusable carbon dioxide. After 20 minutes of slow, deep breathing, with only a small intake of fresh air, his nausea had not developed although he had a slight headache. Twenty minutes later, after further rebreathing, there was no visual disturbance, nausea, or headache. The patient commented that while he was surprised to be so much better, so quickly, the technique had left him feeling as if he had "run up a down escalator."

Case 2—A 29-year-old overweight and bronchitic Post Office worker had a 19-year history of classical migraine. His attacks occurred up to twice a week, lasted about six to eight hours, and were heralded by teichopsia and diplopia, which lasted 10 minutes. At the onset of the headache he developed a right ptosis and his eye would become fully closed if the attack was severe. Using the aura as an indication to begin rebreathing he was able to stop those attacks in which he thought the nausea and pain would not be very intense. On two occasions he persevered with rebreathing for 20 to 30 minutes at the onset of what he considered were the beginnings of severe attacks. On these occasions he lapsed into a state of unconsciousness to awake without a headache or nausea one to two hours later. Because of this disconcerting effect he was reluctant to continue rebreathing for very long.

Cases 3-6—The table gives details of these cases.

Details of cases 3 to 6. Cases 1 and 2 had taken other medication. This consisted of simple analgesics and in case 1 an additional 5 mg of diazepam

Case No	Age (years)	Sex	Age of onset/ migraine type	Usual duration of attack (hours)	Rebreathing time (minutes)	No of attacks stopped	No of attacks rebreathing attempted
3	65	F	Teens common	12	10–15	4	4
4	25	M	19 common	18–24	10–15	2	3
5*	65	F	50 common	24–36	10–15	2	6
6*	65	F	57 common	24–48	15–20	4	8

*Identical twins.

Comment

It has been shown[2] that patients can prevent their migraine attacks from developing by breathing carbon dioxide-oxygen mixtures at the onset of their attacks, but not by breathing carbon dioxide-air mixtures. These studies suggest that oxygen may be important in preventing the development of an attack, although more recently oxygen inhalation has not proved to be effective.

In the series reported here patients rebreathed air. In such a "closed circuit," carbon dioxide pressure must rise and oxygen pressure should fall. Thus in this group of patients the rise in carbon dioxide pressure, or other associated changes, or both, seemed to be important in aborting their migraine attacks. In case 5 unconsciousness induced by rebreathing was probably due to lack of anoxic drive, which is well recognised in some subjects and indeed more common in those with chronic respiratory disease, such as this subject. If carbon dioxide is the active agent in preventing migraine attacks, its mechanism of action is hard to explain. The contemporary view is that the migraine aura is caused by intracranial vasoconstriction and that the headache phase is due to extracranial vasodilatation. Carbon dioxide, a powerful vasodilator, should therefore increase the head pain. Possibly rebreathing protects by reversing the initial vasoconstriction, halting the migraine process. Alternatively, if the concept of two types of migraine sufferers, "dilators" and "constrictors," is correct, rebreathing should benefit the constrictors. Recently, however, it has been suggested[3] that extracranial vasodilatation may not contribute appreciably to migraine pain.

While it is difficult to explain the possible therapeutic effect of rebreathing in migraine, changes induced by hyperventilation[4] in the interictal encephalograms of some subjects with migraine remain unexplained. If the carbon dioxide pressure is important this may explain why exercise and excitement with increased ventilation can provoke attacks and why sleep, perhaps due to reduced ventilation, can cut short attacks. Nevertheless, the beneficial effects of rebreathing may be due not to the raised carbon dioxide pressure but to other changes associated with rebreathing. For instance, it has been postulated[5] that endorphins minimise the stress of chronic airway obstruction; perhaps rebreathing achieves its therapeutic effect in migraine through the release of endorphins secondary to the respiratory distress it causes. Whatever the mechanism by which rebreathing aborts migraine attacks, new light may be thrown by this near-ancient technique on to an even older problem.

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